Appendix E. Public Health Technical Appendix

Appendix E. Public Health Technical Appendix

Introduction

This appendix provides detailed information supporting the analysis presented in Chapter 5, "Public Health". Part 1 describes the potential pathogenic microorganisms that have been known to be present in sewage sludges and provides data on the incidence of reportable diseases in California on a county-by-county basis and for each year for the past 6 to 8 years. Part 2 describes the U.S. Environmental Protection Agency's (EPA's) development of the national sewage sludge regulations (Part 503 regulations). Part 3 provides information on endocrine disruptors, an issue of increasing concern with regard to long-term impacts of chemicals in the environment.

Part 1. Diseases of Interest

This section discusses each of the groups of potential pathogens of concern or specific potential pathogens of concern that may be found in biosolids and summarizes available information on the incidence of diseases they cause in California. This discussion is intended to provide background information for the impact analysis presented in Chapter 5. The information on disease incidence reflects the data collected by the existing statewide voluntary public health reporting system, in which local health departments (two city and all county health departments) participate.

Bacterial Diseases

Enterotoxic *E. coli* 0157

This mutant form of *E. coli* first appeared in the United States in 1982 and is one of hundreds of varieties of *E. coli* found in the guts of mammals (Padhye and Doyle 1992). It is mainly an infection in cattle that can be passed to humans who eat foods contaminated by cattle manure (even in organic gardens using uncomposted manure) or who eat inadequately cooked meat (Cieslak et al. 1992, Centers for Disease Control

1993, Nelson 1997). This particular variety, according to Wells et al. (1991), can be found in 1%–3% of all cattle in the United States but causes them no harm. The infection can be serious for a human host, however, causing severe, often bloody diarrhea. In the worst cases, particularly in young children, *E. coli* can kill. Most often, *E. coli* illnesses are associated with eating undercooked hamburger or uncooked fruits (apples and cantalopes) and vegetables (lettuce in particular) or with person-to-person contact (Belongia et al. 1993, Nelson 1997). Contaminated water supplies are also of growing concern (Jones and Roworth 1996). This particular bacterial strain is of growing concern as more outbreaks occur (Koutkia 1997).

The most well-publicized recent case of illness from *E. coli* is that of three children who died in Washington in 1993 after eating contaminated hamburgers at a fast-food restaurant (Centers for Disease Control 1993). In summer 1997, 25 million pounds of hamburger, potentially tainted with *E. coli* 0157:H7, were recalled by Hudson Foods in Columbus, Nebraska, after consumer illnesses were reported. Illness caused by *E. coli* 0157:H7 has been a reportable disease in California since 1993; the annual number of cases has ranged from 0 to 33, and occasional outbreaks have occurred in major urban areas (Table E-1).

Table E-1. Reported Incidence of Enterotoxic *E. coli* 0157 in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)			1	4	1	
Los Angeles		2	1	14	7	9
Pasadena (City)				1		
San Francisco		1				
Alameda				9		
Marin				1		
San Benito				1		
San Diego				1		
Santa Cruz				1		
Tulare	__	__	__	1	__	__
Total Number of Reported Cases	0	3	2	33	8	9

Source: Starr pers. comm.

Like other pathogens of concern, the enterotoxic form of *E. coli* has a low infectious dose (estimated to be as low as 10 bacteria).

The present detection method for *E. coli* 0157:H7 requires growing the bacteria in laboratory cultures, which takes days. A group of Montana researchers led by Dr.

Gordon McFeters has developed a new method using an antibody test kit. The test takes only 4 hours; is highly sensitive; and works in food, feces, and water. The method could be adapted to detect other foodborne pathogens, such as *Salmonella*, and could be used at various points in beef supply processing to check for contamination.

Campylobacteriosis

Campylobacter jejuni, like *E. coli*, can cause severe cases of gastroenteritis (campylobacteriosis) and has been consistently listed as a pathogen of concern in relation to sludge management (U. S. Environmental Protection Agency 1985) despite a lack of information on its densities in sludges. This pathogen has at times outranked *Salmonella* as a leading cause of bacterial diarrhea (as in 1996), particularly in infants (Table E-2). The reported incidence of gastroenteritis attributable to *C. jejuni* in California has ranged from 864 to 2,477 cases annually since 1993 (Table E-2). Most of the cases (81%) were reported to have occurred in Los Angeles County. No cases were reported in the three counties of the Central Valley where most of the biosolids land application occurs (see Chapter 5). Little has been reported in scientific literature about the levels of this pathogen in feces shed by ill people, its removal in treatment, levels in biosolids, infectious dose, or longevity in the environment (Feachem et al. 1980, U.S. Environmental Protection Agency 1985) as indicated in (Table 5-1 of Chapter 5).

Table E-2. Reported Incidence of Campylobacteriosis in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	73	61	56	93	92	67
Los Angeles	836	765	785	1,149	979	792
Pasadena (City)	15	33	33	12	18	20
San Diego						
San Francisco	6	4	11			
Alameda				537		
Amador				12		
Butte	1					
Calaveras				11		
Colusa				2		
Fresno				15		
Glenn				4		
Imperial				19		
Inyo				6		
Lake				5		
Lassen				4		

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Marin				167		
Mariposa				3		
Mendocino				3		
Modoc				3		
Monterey				2		
Orange		1	29	59	47	24
Placer				1		
Plumas				2		
Sacramento				86		
San Benito				18		
San Bernardino				5		
San Diego				6		
Santa Clara				3		
Santa Cruz				100		
Shasta				9		
Sierra				1		
Siskiyou				13		
Tehama				2		
Trinity				3		
Tulare				115		
Tuolumne				7		
Total Number of Reported Cases	931	864	914	2,477	1,136	903

Source: Starr pers. comm.

Salmonellosis and Typhoid Fever

The bacterial genus *Salmonella* consists of more than 2,000 known serotypes found in different reservoirs and locations, many of which are pathogenic to humans and other animals (Argent et al. 1977, 1981; Ayanwale 1980; Mishu et al. 1994). Ingestion of an infectious dose of *Salmonella* (usually a large number of bacteria is required, as shown in Table 5-1 in Chapter 5) can result in gastroenteritis, enteric fever, and/or septicemia. The two major disease syndromes associated with *Salmonella* are salmonellosis (gastroenteritis) and typhoid fever (enteric fever).

Salmonellosis. The major vehicle of salmonellosis is food (St. Louis et al. 1988, Mishu et al. 1994), although waterborne outbreaks have occurred. There are many zoonotic reservoirs for salmonellosis, including such domestic and wild animals as poultry, swine, cattle, rodents, dogs, cats, turtles, and tortoises. Waterborne outbreaks of salmonellosis occur worldwide and are associated primarily with fresh water.

Salmonellosis is characterized by acute abdominal pain, diarrhea, nausea, fever, and dehydration and is sometimes accompanied by vomiting. The illness can lead to complications and more serious infections. Death is not common except in the very young, the very old, or the debilitated.

It has been estimated that 400,000 to 3.7 million cases (17.3 cases per 100,000) of salmonellosis (including foodborne and waterborne transmission) occur every year in the United States (EOA 1995), with as many as 70% of the cases being imported from foreign travelers. Between 1,010 and 1,894 cases have been reported yearly in California over the past six years (Table E-3), with over 90% of the total being reported in Los Angeles County. No cases were reported to have occurred in those counties in the Central Valley where the highest amounts of biosolids are being land applied.

Recent research on the causes of a *Salmonella* outbreak among chickens has raised concern about the importance of *Salmonella* in wastewater management and indicates the need for constant vigilance and monitoring of the effectiveness of management techniques and disinfection methods (Kinde et al. 1996, 1997). Concern also exists regarding the transmission of *Salmonella* from biosolids to animals (Jones et al. 1980; Argent et al. 1977, 1981) and the ability of the pathogen to survive under hostile environmental conditions (Droffner and Brinton 1995); this ability makes them the indicator of choice for monitoring the effectiveness of biosolids pathogen reduction (U.S. Environmental Protection Agency 1992). In developing the Part 503 regulations, the EPA based its requirements for pathogen reduction and its risk assessments for protection of public health on *Salmonella* because of its high incidence rates, its ability to regrow, and its correlation with coliform bacteria (about 1.4 *S. typhi* per million coliforms based on a morbidity rate of 0.18/million persons).

Typhoid Fever. Typhoid is transmitted via water or food contaminated by the feces or urine of a carrier. Fruits, vegetables, and milk contaminated by sewage or by the hands of carriers are also modes of transmission. The case-fatality rate for typhoid fever can reach 10% if symptoms go untreated; there are approximately 500 fatalities per year (0.2 per 100,000 deaths per year) in the United States.

Table E-3. Reported Incidence of Salmonellosis in California
(1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	88	107	27	104	100	82
Los Angeles	1,034	1,348	1,208	1,152	1,112	881
Pasadena (City)	29	37	27		33	17
San Diego	1					
San Francisco	1	6	1			
Alameda				280		
Amador				3		
Calaveras				5		
Colusa				3		
Contra Costa				1		
El Dorado				5		
Fresno				7		
Glenn				6		
Imperial				39		1
Inyo				6		
Lake			1	7		
Lassen				4		
Marin				35		
Mariposa				2		
Mendocino				1		
Modoc				1		
Mono				16		
Orange			47	37	47	28
Placer				4		
Plumas				6		
Sacramento				2		
San Benito				7		
San Bernardino				4		
San Diego				3		1
San Luis Obispo				1		
Santa Barbara				1		
Santa Clara				2		
Santa Cruz				60		
Shasta				6		

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Tehama				5		
Tulare				68		
Tuolumne				11		
Total Number of Reported Cases	1,153	1,498	1,311	1,894	1,292	1,010

Source: Starr pers. comm.

Shigellosis

The genus *Shigella* is made up of four species of rod-shaped bacteria that are all pathogenic in humans and other primates. The four species are characterized as groups or types: Group A, *S. dysenteriae* (10 serovars); Group B, *S. flexneri* (17 serovars); Group C, *S. boydii* (15 serovars); and Group D, *S. sonnei* (1 serovar). Shigellosis, an acute bacterial disease caused by *Shigella*, occurs worldwide, with outbreaks common under conditions of crowding and poor sanitation (i.e., jails, institutions for children, mental hospitals, crowded camps and ships). The reporting for the disease distinguishes between the four groups to help identify the sources and potential severity of the infection. From 1967 to 1988, annual isolation rates of *Shigella* reported to the Centers for Disease Control (CDC) varied between about 5 and 10 per 100,000 persons. It has been estimated that 5% of all symptomatic cases of shigellosis are reported to the national surveillance system. *Shigella* is considered the most highly communicable of the bacterial diarrheas; as few as 10 organisms have been reported to cause clinical illness (U. S. Environmental Protection Agency 1985).

For *S. dysenteriae* (Shiga bacillus) infection, case-fatality rates approach 20%; for *S. sonnei infection*, the infection is short-lived and the fatality rate is almost negligible, except in immunocompromised persons. Few cases are reported in California. The annual number of cases reported in the state ranges from 0 to 17 cases a year for Group A, 196 to 796 for Group B, 2 to 45 for Group C, and 388 to 873 for Group D (Tables E-4, E-5, E-6, and E-7, respectively). Some 62–178 cases a year were unidentified as to type (Table E-8). Overall, some 701 to 1,530 cases per year have been reported from 1993 to 1998. None of these cases has been associated with biosolids.

Shigella spp. has in the past been the most common bacterial pathogen implicated in waterborne outbreaks in the United States, but its occurrence has declined over time (Moore et al. 1993). Shigellosis also has been implicated in outbreaks associated with recreational swimming (Blostein 1991, Sorvillo et al. 1988).

Shigellosis is transmitted via the fecal-oral route, directly or indirectly, primarily from person to person via contaminated food and water. In areas of poor sanitation, food and water may play a greater role in transmission. Flies have been shown to be a vector in the transmission of the disease (Dunaway et al. 1983).

The survival of *Shigella* in water, soils, and plants depends on factors such as temperature and the concentration of other bacteria, nutrients, and oxygen. In various studies, *Shigella* has been shown to survive for up to 22 days in well water and even longer in colder temperatures (47 days) and up to 135 days in permafrost soils of Siberia (EOA 1995).

One detailed review of the scientific literature performed by EOA (1995) found no *Shigella* outbreaks associated with water where the source met the coliform standards at the time of exposure.

Table E-4. Reported Incidence of Shigellosis Type A in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)			1	1		
Los Angeles	14	7	3	9	0	5
Pasadena (City)		1	1			
Alameda				3		
Lassen				1		
Marin				1		
Santa Cruz				1		
Shasta				1		
Total Number of Reported Cases	14	8	5	17	0	5

Source: Starr pers. comm.

Table E-5. Reported Incidence of Shigellosis Type B in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	36	46	28	29	26	19
Los Angeles	398	363	352	284	215	171
Pasadena (City)	3	9	5	6	4	4
San Francisco	2	378	2			
Alameda			27			
Colusa			1			
Fresno			1			
Imperial			7			
Inyo			1			
Marin				5		

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Mono				1		
Orange			11	4	6	2
San Benito				10		
San Bernardino				2		
Santa Cruz				3		
Tulare				4		
Total Number of Reported Cases	439	796	435	348	251	196

Source: Starr pers. comm.

Table E-6. Reported Incidence of Shigellosis Type C in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	1	2		3	2	2
Los Angeles	28		44	17	28	20
Pasadena (City)				2		
Alameda				3		
Colusa				1		
Imperial				1		
Lassen				1		
Orange			1			1
San Benito				4		
Total Number of Reported Cases	29	2	45	32	30	23

Source: Starr pers. comm.

Table E-7. Reported Incidence of Shigellosis Type D in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	102	30	64	46	61	98
Los Angeles	578	399	652	510	306	292
Pasadena (City)		38	35	16	9	7
San Francisco	1	1	5			
Alameda			89			
Fresno			17			

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Imperial			11			
Marin				4		
Orange				6	12	
San Benito				5		
San Bernardino				1		
San Diego		1		3		
Santa Cruz				15		
Shasta				1		
Tulare				18		
Ventura	1					
Total Number of Reported Cases	682	469	873	625	388	397

Source: Starr pers. comm.

Table E-8. Reported Incidence of Shigellosis (Unidentified as to Type) in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	8	3	5	4		3
Los Angeles	108	101	137	103	60	77
Pasadena (City)			1	1	2	
Alameda			28			
El Dorado				2		
Imperial				24		
Marin				5		
Modoc				2		
Orange		1	1			
San Benito				1		
San Diego				2		
Santa Cruz				9		
Shasta				1		
Tehama				1		
Tulare				23		
Total Number of Reported Cases	116	105	172	178	62	80

Source: Starr pers. comm.

Protozoan Diseases

Amoebiasis

Amoebiasis, an infection caused by the environmentally resistant pathogen *Entamoeba histolytica*, is acquired by mouth contact. Symptoms can vary from minor abdominal cramps to severe diarrhea alternating with constipation. The incidence of disease from this protozoan is low; between 127 and 237 cases per year have been reported in California over the past six years (Table E-9). None of the reported cases have been associated with biosolids or wastewater management. Over 94% of the reported cases in California were in Los Angeles County (including Long Beach and Pasadena), reflecting the size of the population. This disease is associated often with travel in other countries, particularly in areas of Mexico.

Table E-9. Reported Incidence of Amoebiasis in California
(1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	21	14	13	16	13	14
Los Angeles	213	156	145	161	110	113
Pasadena (City)	2			2		
San Francisco	1	5	5		2	
Marin				30		
Mariposa				1		
Orange				3		
Sacramento				6		
Santa Clara				2		
Santa Cruz						
Tehama				1		
Tulare	—	—	—	1	—	—
Total Number of Reported Cases	237	175	163	223	125	127

Source: Starr pers. comm.

Crytosporidiosis

Cryptosporidiosis is a gastrointestinal infection that is caused by the protozoan *Cryptosporidium* spp. *Cryptosporidium* oocysts are shed by humans and animals in feces. The infectious dose in humans is thought to be small; it is 10–400 oocysts in species other than humans. Little is known about the concentrations of viable oocysts in biosolids (Gerba pers. comm.) and the viability of oocysts in the environment, but oocysts are known to have the potential to survive months following their excretion (EOA 1995) and have the potential to survive more than a month following sludge treatment and land application (Whitmore and Robertson 1995). However, it has been found that conventional treatment and anaerboic digestion are effective in reducing the numbers of oocsysts in biosolids (Whitmore and Robertson 1995).

Modes of transmission for cryptosporidiosis include person-to-person contact, zoonotic transmission, and contaminated food and water. Person-to-person transmission is probably the most important mode and has been documented among family/household members, sexual partners, health workers and their patients, and children in day care centers. *Cryptosporidium* readily crosses host-species barriers as well, though, and human infections are often the result of zoonotic transmission. *Cryptosporidium* is harbored by more than 40 mammals. Reservoir hosts include calves, dogs, cats and rodents (Tzipori 1988).

Several waterborne outbreaks of cryptosporidiosis have been reported in the United States where the filtration component of water treatment was suboptimal (Milwaukee, for example - see below) (McKenzie et al. 1994). Cryptosporidiosis also has been associated with recreational use of swimming pools (Joce et al. 1991). Disease incidence in England associated with chlorinated water supplies and swimming pools indicates cryptosporidiosis resistance to chlorination (Furtado et al. 1998).

During a waterborne outbreak of cryptosporidiosis resulting from contamination of a public water supply that affected an estimated 13,000 people in Georgia, routine samples from the water system were found to meet EPA and State of Georgia standards for coliform bacteria (Robertson and Smith 1992). During another cryptosporidiosis outbreak associated with public water supply that led to an estimated 403,000 cases of diarrhea in Milwaukee, coliforms were not detected in samples of treated water (McKenzie et al. 1994). It should be noted that it is generally recognized that *Cryptosporidium* oocysts are removed or inactivated by effective and reliable water treatment practices where the water supply is not contaminated by dairy or pasture runoff (most often from flooding).

Cryptosporidium is found worldwide. Human cryptosporidiosis has been reported in at least 60 countries on six continents, with widely varying prevalence among those seeking medical care for diarrhea (EOA 1995). The prevalence is highest in non-industrialized regions: Europe,1% to 2%; North America, 0.6% to 4.3%; and Asia, Australia, Africa, and Central and South America, 3% to 20%. Seroprevalence rates in immunocompetent individuals are between 25% and 35% in the United States and are well over 50% in Latin America. Children generally have a significantly higher prevalence than adults, and infections are often seasonal, with a higher prevalence during warmer, wetter months.

No outbreaks associated with biosolids use have been reported in scientific literature or with the health agencies consulted during the preparation of this EIR. This disease is rare, with 31 to 212 cases a year reported in California, none of which are from areas where biosolids have been land applied (Tables E-10 and E-11).

Table E-10. Reported Incidence of Cryptosporidiosis in California (1991 through 1998)

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Long Beach (City)		3	18	4	24	10	4	5
Los Angeles		56	68	145	171	144	57	68
Pasadena (City)				4	1	2	1	1
San Diego								
San Francisco	16		3	1	2			
Fresno						1		
Kern	1							
Marin		2				1		
Orange	3	2	1	1	1	2		1
Riverside						1		
Sacramento						1		
San Bernardino						2		
San Diego						2		
San Luis Obispo	1							
San Mateo	1							
Sonoma	1							
Total Number of Reported Cases	23	63	90	155	199	166	62	75

Source: Starr pers. comm.

Table E-11. Reported Incidence of Cryptosporidiosis (Type S) in California (1991 through 1998)

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Long Beach (City)	1		4	1	1	2		
Los Angeles	3	41	41	17	10		38	16
Pasadena (City)			2		1		1	
Alameda						1		
Amador								
Monterey		4						
Napa								
Orange	1		3		1		3	
San Bernardino	1							
San Diego	1							
Santa Clara	1							
Total Number of Reported Cases	8	45	50	18	13	3	42	16

Source: Starr pers. comm.

Giardiasis

Giardia lamblia is a protozoan that principally infects the upper small intestine in humans, who can often be asymptomatic. *Giardia* infection, or giardiasis, manifests itself in the form of chronic diarrhea, abdominal cramps, weight loss, and fatigue that can last for months with relapses. It can progress to cause malabsorption syndrome, in which digestion is impaired and weight loss occurs. Certain immunodeficiency syndromes also may be associated with *Giardia* infection, and the infection is particularly devastating in immunocompromised persons. Carriers can shed *Giardia* for years, but a self-cure usually occurs within 2 to 3 months. The numbers of *Giardia* cysts shed in feces are highly variable but have been measured to be as high as 900 million per day (Feachem et al. 1983).

Before leaving the intestine, *Giardia* generally forms a resistant cyst, which is highly resistant to traditional disinfection techniques (EOA 1995). The cysts can remain viable in water for several months and can remain viable in soils as well, but cannot tolerate freezing (EOA 1995). It has been found that the presence of traditional bacterial indicators does not correlate with the presence of cysts, particularly in unfiltered but disinfected drinking water (EOA 1995). Negative coliform tests do not provide assurance that water is free of *Giardia* cysts; however, positive coliform results often correlate with *Giardia* outbreaks (EOA 1995).

The major reservoir of *Giardia* is humans, but there is evidence that humans may acquire infections from other animals. Beavers may be a reservoir and have been implicated in waterborne outbreaks (EOA 1995). Dogs, gerbils, guinea pigs, beavers, raccoons, bighorn sheep, and muskrats have all been shown to be carriers of *Giardia* (EOA 1995).

Giardia infection is transmitted through contaminated water supplies, foodborne outbreaks, and person-to-person contact, with the later being the most prevalent means of transmission. Individuals with impaired immune function appear to have increased susceptibility to *Giardia* infection.

The numbers of *Giardia* cysts in biosolids have been estimated to range from 10 to 10^3 per gram with no removal via treatment. However, significant viability reduction occurs during digestion, estimated in laboratory studies to be as high as 99.9% inactivation (Straub et al. 1993, Cravaghan et al. 1993). Class A treatment requires that treated biosolids contain less than one protozoan cyst per gram. For Class B sludge generated in Australia, it has been found that anaerobically digested and mechanically dewatered sludge had cysts present at levels of public health concern after 1 year, but that cysts were destroyed after only 12 weeks following soil amendment (Hu et al. 1996).

Giardia is found worldwide. The prevalence of *Giardia* infection worldwide has been estimated to be about 7%, and infection is more common in children than adults. Prevalence rates vary between less than 1% and 50% and depend on the population sampled, infection rates being highly dependent upon sanitation and the quality of drinking water. Areas of the United States known to be associated with increased risk of infection are usually mountainous and include New England, the Pacific Northwest, and the Rocky Mountains.

The number of cases reported in California is variable, ranging from 510 to 1,335 per year (Table 5-6 in Chapter 5). The incidence in California is the highest in Los Angeles County, where more than 88% of the cases were reported. No cases were reported in Kern, Merced, and Kings Counties, where the majority of the biosolids application currently occurs (Table E-12). No cases of the illness associated with biosolids operations have been reported (Cook and Shaw pers. comms.).

Table E-12. Reported Incidence of Giardiasis in California (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	88	89	70	85	276	63
Los Angeles	971	700	588	618	528	427
Pasadena (City)	28	25	12	26	22	20
San Diego	1	1				
San Francisco	1	5	3			
Alameda				152		
Amador				8		
Calaveras				12		
Colusa				2		
Contra Costa				1		
El Dorado				1		
Fresno				21		
Glenn				5		
Imperial				10		
Inyo						
Lake				14		
Lassen				5		
Marin				75		
Mariposa				2		
Mendocino				2		
Modoc				1		
Mono				1		
Orange		1	19	125	32	
Placer				2		
Plumas				4		
Sacramento				63		
San Benito				6		
San Bernardino				5		
San Diego				4		
San Luis Obispo						
Santa Barbara			1			
Santa Clara				1		
Santa Cruz						
Shasta				4		

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Sierra				1		
Siskiyou				3		
Tehama				9		
Trinity				3		
Tulare				59		
Tuolumne				5		
Total Number of Reported Cases	1,089	821	693	1,335	858	510

Source: Starr pers. comm.

Viruses

Hepatitis A

The hepatitis A virus (HAV) is a virus physically resembling an enterovirus that causes hepatitis A, an illness with the symptoms of fever, nausea, malaise, anorexia, and abdominal discomfort, followed by jaundice. The disease can be mild, lasting 1 to 2 weeks, or severe, with disabling effects lasting months in rare cases. The recovery period is usually prolonged. The case-fatality rate has been reported to range from 0.04% in children 5–14 years old to 2.7% in adults over 49 years old, with typical case-fatality rates of 0.1–0.5%. Relapse rates can be as high as 20%. Hepatitis A can be diagnosed by the detection of virus in the stool or the presence of IgM antibodies against HAV in the serum of persons who are acutely ill. There is currently no specific treatment for HAV.

The normal reservoir of HAV is acute-phase humans; there is no known carrier state. Mode of transmission is via the fecal-oral route, with person-to-person transmission being the most frequent means of transmission, usually via water or food. HAV can survive for long periods on inanimate objects and on human hands; therefore, food contamination by infected persons is a major area of concern. In the United States, waterborne outbreaks have been estimated to contribute 0.4%–8% of all HAV incidence, and no waterborne disease outbreaks have been shown to have been directly associated with biosolids. The majority of waterborne outbreaks in the United States involve small private or semiprivate water supplies with or without chlorination; these outbreaks are usually attributable to plumbing-sewage cross-contamination or to a raw-water source being so grossly polluted with sewage that virus levels cannot be eliminated by treatment of the water using conventional methods. The infectious dose is estimated to be in the range of 1 to 10 plaque-forming units (PFUs).

Little is known about persistence of hepatitis A in the environment. Survival in water has been recorded for as long as 40 days in surface waters and 70 days in groundwaters

(EOA 1995). Levels in biosolids have not been reported in anaerobically digested sludge.

There is no known direct correlation between HAV and indicator organisms such as coliform bacteria, fecal streptococci, acid-fast bacteria, or coliphage.

Hepatitis A has a worldwide distribution. Since 1920 in the United States, there have been about 15 reported outbreaks of HAV associated with drinking water, most of which are reported from areas with poor sanitation or contaminated water supplies (Singh et al. 1998). In California, the number of Hepatitis A cases has ranged from 474 to 1,415 annually over the past eight years (Table E-13).

Incidences in counties where biosolids are being land applied have not increased since land application was intensified in recent years, and no cases have been reported in most instances in the past seven years. None of the cases reported can be related to the handling or use of biosolids.

Table E-13. Reported Incidence of Hepatitis A in California
(1991 through 1998)

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Long Beach (City)	11	2	93	122	207	198	168	73
Los Angeles	100	1,005	707	733	760	801	1,209	619
Pasadena (City)	5	1	38	37	15	19	21	14
San Francisco	41		14	37	59			
Alameda	14	6				36		
Amador						3		
Butte						1		
Colusa						4		
Contra Costa	7					1		
El Dorado	6	3		2		1		
Fresno	8					10		
Glenn						3		
Humboldt	6							
Imperial	1					26		
Kern	19							
Kings	4							
Lake	3							

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Lassen						1		
Madera		1						
Marin		3				17		
Mendocino	2	4				2		
Merced	6	8						
Modoc						1		
Mono		1						
Monterey	12	3						
Napa	1	1						
Orange	35	3	22	22	37	25	17	19
Placer	1	4						
Riverside	18	9			1	3		
Sacramento	11					27		
San Bernardino	25					21		
San Diego	61					18		
San Joaquin	1							
San Luis Obispo	1							
San Mateo	8							
Santa Barbara	2							
Santa Clara	17					2		
Santa Cruz	2					11		
Shasta	2					11		
Siskiyou	2					2		
Solano	1							
Sonoma	7							
Stanislaus	9							
Sutter	1							
Tehama						3		
Trinity						1		
Tulare	14					51		
Tuolumne	1							
Ventura	6							
Yolo	1							
Yuba	2					1		
Total Number of Reported Cases	474	1,054	874	953	1,079	1,300	1,415	725

____

Source: Starr pers. comm.

Viral Meningitis

"Viral meningitis" is the general term that refers to all serious viral diseases (not gastroenteritis of unknown origin) that have been reported. Included as causative agents and reportable as viral meningitis are the Coxsackievirus A and B, Echovirus, and new enteroviruses (acquired orally). It is unknown how many viruses cause gastroenteristis and flulike symptoms that are unreported. The reportable cases of viral infections have ranged from 119 to 485 per year (Table E-14). Most of the cases are reported in the more urbanized counties and the numbers of reported cases are largely proportional to population. Only two cases have been reported in the three largest land application areas, both in Kern County. There is no evidence that any of the cases are associated with biosolids land application operations.

Table E-14. Reported Incidence of Viral Meningitis in California (1991 through 1998)

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Long Beach (City)	3	36	69	18	22	35	30	87
Los Angeles	13	413	317	155	89	105	134	276
Pasadena (City)			8	3	1	3	3	10
San Diego	40					6		
Alameda	1					2		
Contra Costa	4							
Fresno	9					4		
Glenn						1		
Imperial	3							
Kern	2							
Lassen						2		
Marin		5				2		
Mendocino		1						
Monterey	2	1						
Napa	2	1						
Orange	62	23	30	5	7	11	19	30
Placer		1						
Riverside	20	2						
Sacramento	7		1			3		
San Bernardino	10					1		
San Joaquin	1							
San Luis Obispo	1							
San Mateo	3							
Santa Clara	3							

Location by County/City Health Department	1991	1992	1993	1994	1995	1996	1997	1998
Santa Cruz	3					8		
Solano	1							
Sonoma	1							
Tulare	4					5		
Tuolumne	1							
Ventura	1	2						
Yuba	1							
Total Number of Reported Cases	198	485	425	181	119	188	186	403

Source: Starr pers. comm.

Gastroenteritis

Gastroenteritis is a widespread disease that can be caused by numerous known and unknown viral agents. Person-to-person transmission is the principal mechanism for the spread of many infections; therefore, the most important element in preventing and controlling outbreaks is improved environmental hygiene (i.e., food, water, and sanitation).

When foods other than shellfish are implicated in viral gastroenteritis outbreaks, the contamination has usually taken place near the point of consumption (shellfish are not discussed in this EIR because of the nature of the project). Ill food handlers were identified in nine of the 15 documented Norwalk outbreaks reported to the CDC from 1985 to 1988 for which adequate epidemiologic data were available (Centers for Disease Control unpublished data). Foods that require handling and no subsequent cooking (e.g., salads) constitute the greatest risk. Among Norwalk-confirmed foodborne outbreaks from 1976 to 1980 that were not attributable to shellfish, salad was the most commonly implicated food (Centers for Disease Control 1999).

The long list of foods implicated in outbreaks of viral gastroenteritis reflects the variety of foods handled by food-service personnel and the low infectious dose (10–100 particles) of most viral agents of gastroenteritis. In contrast to the factors important in amplifying bacterial contamination, practices such as leaving foods unrefrigerated or warming them for prolonged periods are not direct risk factors for increased viral transmission because the viruses do not multiply outside the human host.

The Norwalk agent can remain infective even if frozen for years or heated to 60EC for 30 minutes. Cooking temperatures at 100EC or above are probably adequate to inactivate Norwalk and most other enteric viral pathogens.

Outbreaks of viral gastroenteritis have been associated with various sources of contaminated water, including municipal water, well water, stream water, commercial ice, lake water, and pool water (Centers for Disease Control 1999). Disinfection of municipal supplies may not be adequate to kill the Norwalk agent, which can remain highly infective despite 30-minute exposure to concentrations of chlorine as high as 6.25 milligrams per liter (mg/l) and levels of 10 mg/l (Centers for Disease Control 1999); this helps explain why this virus is predominant in waterborne disease outbreaks. Rotavirus, for which only one waterborne outbreak has been documented in the United States, is more sensitive to chlorine than the Norwalk agent.

Because rotaviruses can survive for several days on nonporous materials in conditions of low temperature and humidity, objects may contribute to their transmission. A recent study of a Norwalk viral outbreak on a cruise ship implicated toilets shared between staterooms as a risk factor for infection, suggesting that surfaces contaminated by Norwalk particles from spattered or aerosolized material may play a role in transmission of Norwalk-like viruses causing gastroenteritis.

Aerosolized rotavirus has also been observed to cause~~d~~ diarrheal illness in experimental mice. Studies are needed to address the efficacy of barrier precautions (e.g., face shields, respirators) in interrupting transmission of these agents (Centers for Disease Control 1999).

Contaminated hands (hands contaminated directly or through contact with contaminated surfaces) may be the most important means by which enteric viruses are transmitted; thus, any people involved with biosolids should avail themselves of handwashing with soap on a routine basis to control the spread of all enteric pathogens.

Nearly all the agents of viral gastroenteritis in humans have related strains that can cause diarrhea in animal species. These strains appear to be highly host-specific, however, and zoonotic transmission has not been documented as having an important role in human disease, either endemically or in outbreaks.

Acquired Immune Deficiency Syndrome (AIDS/HIV Virus)

No discussion of viruses would be complete without a discussion of acquired immune deficiency syndrome (AIDS), which is caused by HIV (human immunodeficiency virus). It is noteworthy that HIV has never been recovered from wastewater samples into which it has not been artificially introduced (Ansari et al. 1992, Casson et al. 1992, Moore 1993). Researchers have recovered viral nucleic acid fragments in wastewater but none in biosolids (Preston et al. 1991). However, the detection of nucleic acid sequences does not represent the presence of viable HIV. No intact HIV has been recovered from either raw sewage or biosolids. The CDC contends that wastewater treatment professionals, as well as members of the public who may contact wastewater or biosolids, are not at risk of contracting AIDS as a result of this contact (Centers for Disease Control 1999).

Parasitic Worms

Several parasitic intestinal worms are found in wastewater (Straub et al. 1993, ABT Associates 1993). These parasites are a potential hazard to the public health in general and to treatment plant and biosolids workers in particular. The beef tapeworm (*Taenia saginata*) can cause taeniasis if ingested with poorly cooked meat. Tapeworm eggs are detectable in biosolids, but there is no evidence that they have contributed to distribution of the disease except in one reported case discussed below.

Toxoplasmosis

Toxoplasmosis is a very rare disease that affects only unborn fetuses. The disease is derived from cat feces. As shown in Table E-15, between 9 and 42 cases per year have been reported in California, none of which were in areas where biosolids are being extensively land applied. All cases but one were in Los Angeles County; the exception was in San Diego County.

Table E-15. Reported Incidence of Toxoplasmosis (1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Long Beach (City)	2		1	1	1	1
Los Angeles	40	9	27	22	15	8
Pasadena (City)					1	
San Diego	_	_	_	_	1	_
Total Number of Reported Cases	42	9	28	23	18	9

Source: Starr pers. comm.

Roundworms

Ascariasis is caused by the presence of roundworms (*Ascaris lambricoides*) in the intestinal tract. The disease results from the ingestion of roundworm eggs, which survive for months to years in biosolids (Table 5-1 in Chapter 5) and were a primary focus of the EPA Part 503 regulation risk management practices. This disease is rare and is not reported.

Hookworms

Hookworm disease, rare in California but still present in the southeastern United States, is generally acquired when the larvae of *Necator americanus* enter through the bare skin, usually the feet. Infections also have occurred following ingestion of foods contaminated by wastewater. No cases of transmission related to biosolids land application have been reported. Symptoms include malnutrition, loss of energy, and anemia. This disease is rare and has not been reported in the past 6 years.

Tapeworms

There are two species of tapeworms (*Taenia saginata* [beef] and *T. solium* [pork]) that live in the intestinal tract, where they can cause abdominal pain, weight loss, and digestive disturbances (Straub et al. 1993). Humans serve as the definitive host for the adults, and the eggs, which are passed in feces, may not be completely destroyed by all sludge treatment processes (Feachem et al. 1983), thus leading to the potential for their application to land in biosolids. If cattle graze on this land and ingest viable larvae, the disease may be transmitted to cattle. Humans have to become infected from eating incompletely cooked meat containing the larval stage of the tapeworm. A single recorded case of beef tapeworm transmission through the fertilization of land with untreated sludge has been reported in the United States; this case was reported more than 20 years ago, however, before the development of the Part 503 regulations and the improvements in treatment mandated under the Clean Water Act (Hammerberg et al. 1978).

Tapeworm infections are relatively rare in California; a maximum of 14 case per year have been reported, all in Los Angeles County (Table E-16).

Table E-16. Reported Incidence of Tapeworm (*Taenia* sp.)
(1993 through 1998)

Location by County/City Health Department	1993	1994	1995	1996	1997	1998
Los Angeles	2	6	4		1	13
Pasadena (City)	_	_	1	_	_	1
Total Number of Reported Cases	2	6	5	0	1	14

Source: Starr pers. comm.

Fungal Diseases

Fungal pathogens include several species that have been identified in biosolids, as listed below.

Fungal Species	Disease
Aspergillus fumigatus	Aspergillosis
Candida albicans	Candidiasis
Cryptococcus neoformans	Subacute chronic meningitis
Epidermophton spp. and *Trichophyton* spp.	Ringworm and athlete's foot
Trichosporon spp.	Infection of hair follicles
Phialophora spp.	Deep tissue infections

Most of these fungal species have been found associated with composting operations, where they are enhanced by the favorable conditions created (wood chips and heat).

Aspergillosis is illness caused by the *Aspergillus* fungus, which is found commonly growing on dead leaves, stored grain, compost piles, or other decaying vegetation. The fungus can cause illness in three ways: as an allergic reaction in people with asthma (pulmonary aspergillosis, allergic bronchopulmonary type); as a colonization in an old lung cavity that has healed from previous disease such as tuberculosis or in a lung abscess, where it produces a fungus ball called aspergilloma; and as an invasive infection with pneumonia that is spread to other parts of the body by the blood stream (pulmonary aspergillosis; invasive type). The invasive infection can affect the eye, causing blindness, and any other organ of the body, but especially the heart, lungs, brain, and kidneys. The third form occurs almost exclusively in people whose immune systems are suppressed by high doses of cortisone drugs, chemotherapy, or a disease that reduces the number of normal white blood cells. Those at risk include organ transplant recipients and people with cancer, AIDS, or leukemia (Rosenberg and Minimato 1996).

The A*spergillus* group of fungi is generally less prevalent than other fungal species, but it can be pathogenic to people under conditions of high exposure. Normal background levels of *Aspergillus fumigatus* outdoors rarely exceed 150 spores per cubic meter.

Composting facilities do represent sites where there occurs a massive culturing of *Aspergillus fumigatus* organisms in relatively small areas compared with most “natural” or background circumstances. Studies have found concentrations of *A. fumigatus* 10 times higher than background levels in active commercial composting facilities, but the concentrations fell off sharply within 500 feet of the operational site (Clark et al. 1983) If the nearest human receptor is beyond the point at which concentrations fall to background levels, no elevated exposure is occurring.

The use of bark or wood chips (e.g., as a bulking agent for sewage sludge composting) typically raises the onsite level of airborne *A. fumigatus* spores (Millner et al. 1977, 1980; Clark et al. 1983). In one study in Maryland, *A. fumigatus* levels in sewage sludge rose from 10^2 or 10^3 colony forming units per gram dry weight (CFU/gm dry wt) to 2.6×10^6 to 6.10×10^7 CFU/gm dry wt when mixed with wood chips that were stockpiled for various lengths of time. The increase appeared to be caused by wood chips being stored in moist piles that were allowed to generate heat (Millner et al. 1977).

Increased *A. fumigatus* spore concentrations have been observed also in screened compost; the concentrations may have been increased as a result of reinoculation by spores as compost passed through contaminated screens multiple times (Olver 1979); others have suggested that multiple screenings may break up spore clusters, causing more spores to be released.

Numerous researchers (Raper and Fennel 1965; Sinski 1975; Olver 1979; Epstein and Epstein 1985, 1989; Maritato et al. 1992; Epstein 1993) have presented persuasive arguments regarding the lack of health risk from *A. fumigatus* for certain outdoor workplace environments. In enclosed compost facilities without dust control, there is an elevated risk of worker exposure to spores. In a worst-case scenario, a respiratory model developed by Boutin et al. (1987) estimated that a completely unprotected worker shoveling mature compost at a highly contaminated site could inhale 25,000 to 30,000 viable spores per hour. However, elevated exposure is not automatically synonymous with an elevated health risk for compost workers (or neighboring communities). Epstein (1993) discusses several composting facilities in the United States in which health monitoring (physical examinations) of compost workers has been conducted; the results of the physical examinations did not reveal any illnesses directly associated with composting.

Many public health specialists, scientists, and engineers in North America and Europe believe that properly operated composting and co-composting operations present little health risk to normal compost facility employees and present a negligible risk or no risk to nearby residences (Millner et al. 1977, Clark et al. 1983, Epstein and Epstein 1985, Boutin et al. 1987, Maritato et al. 1992). Diaz et al. (1992) stated:

> The existence of hazard from the spores of *A. fumigatus* [at commercial composting facilities] is yet to be demonstrated. The infectivity of the spores is low. Consequently, any danger posed by it would be of significance only to the unusually susceptible individual. Nevertheless, use of respirators by workers and the siting of such facilities in areas remote from residential dwellings and areas where potentially sensitive receptors work of live is warranted as a prudent land use planning practice.

Reducing the dispersal of *A. fumigatus* spores appears to be the best way to reduce exposure and help protect the health of compost workers and the neighboring communities. The following management practices can help reduce the dispersal of spores into the air during commercial aerobic composting operations (whether they involve windrows, aerated static piles, or the various types of in-vessel reactors—vertical, horizontal, or rotating drum):

- suitable siting, design, and construction (berms, vegetation, etc.) of composting facilities;
- implementation of facility operational practices such as dust suppression, modification of time of operation, etc.);
- engineering and administrative controls (enclosed cabs, use of amendment materials, health checks for workers); and
- use of personal protective equipment (respirators or protective masks).

The California Integrated Waste Management Board's current green waste composting regulations require a setback of at least 300 feet of the facility's active compost materials areas from any residence, school, or hospital, excluding onsite residences, unless a variance is granted from the local enforcement agency. More stringent requirements can be applied where there are sensitive receptors; high winds; or other factors related to health risks, such as the health status of the community potentially affected.

Pathogens of Emerging Concern

Research techniques continue to be developed for determining the pathogenic microorganisms responsible for human and animal disease outbreaks. New genetic techniques and electron microscopy have improved our ability to detect and identify pathogens, particularly new viruses. Because approximately 50% of all cases of gastroenteritis are of unknown origin, such research is vital to development of our understanding of disease and disease prevention.

This section describes the results of a literature review of recent outbreaks of disease (worldwide) undertaken to identify some of the emerging pathogens and their possible modes of transmission. The results of this search are summarized in Tables E-17 and E-18 for bacteria and viruses, respectively. Table E-19 provides information on parasites. None of these potential pathogens of concern have yet been identified with the use or handling of biosolids. Most outbreaks are associated with poor sanitation or food preparation and handling or drinking of contaminated water.

The patterns of incidence and pathways of spread for various pathogens are poorly understood. Epidemiological studies have revealed some interesting findings with regard to crytposporidiosis that show how incidence of disease and causative factors are difficult to identify: evaluation of health records and water treatment plant records revealed that outbreaks of cryptosporidiosis were occurring in Milwaukee for more than a year before the large documented outbreak in 1993 (when high runoff occurred, the water treatment plant turbidity levels became very high, and treatment levels declined) (Morris et al. 1998).

Table E-17. Bacterial Pathogens of Emerging Concern

Pathogen	Disease	Source	Environmental Sources	Outbreaks Reported	Literature
Aeromonas spp. (332 types)	Gastroenteritis	Pigs, chickens, ground beef, human feces, fish, milk, vegetables	Drinking water, fresh water, and wastewater	None from biosolids	Wadstrom and Ljungh 1991, Hanninen and Siitonen 1995
Pleisomonas shigelloides	Gastroenteritis	Seafoods	Contaminated seawater	None from biosolids	Wadstrom and Ljungh 1991
Hepatitis E	Hepatitis	Human feces	Sewage-contaminated water supply	None from biosolids; water related only.	Singh et al. 1998
Helicobacter sp.	Unknown	Wastewater, treated water, well water	Contaminated supplies	None from biosolids	Hulten et al. 1998
Salmonella enteritidis PT6	Salmonellosis	Eggs	Foodborne contamination	None from biosolids	Evans 1998, St. Louis et al. 1988, Mishu et al. 1994
Salmonella enteritidis PT4	Salmonellosis	Wastewater to mice to chickens	Treated secondary effluent discharged to surface water	None from biosolids	Kinde et al. 1996, Kinde et al. 1997

Table E-18. Viral Pathogens of Emerging Concern

Pathogen	Disease	Source	Environmental Sources	Outbreaks Reported	Literature
Adenoviruses 40 and 41	Gastroenteritis	Humans	Unknown	None from biosolids	Enriques et al. 1995
Human torovirus	Gastroenteritis and diarrhea	Children	Unknown	None from biosolids	Jamieson et al. 1998
Picobirnavirus	Diarrhea	Adults and children, chickens, rabbits	Unknown	None from biosolids	Cascio et al. 1996; Chandra 1997; Ludert et al. 1995; Gallimore et al. 1995a, 1995b
Coxsachieviruses (new serotypes)	Association with diabetes mellitus	Children	Fecal-oral contact	None from biosolids	Roivainen et al. 1998
Small round structured virus (SRSV)	Influenza	Infants, children, elderly	Unknown	None from biosolids	Dedman et al. 1998
Norwalk-like virus (calicivirus)	Unknown	Pigs	Unknown	None from biosolids	Sugieda et al. 1998
Swine HEV (hepatitis E virus in pigs)	Unknown	Pigs	Unknown	None from biosolids	Meng et al. 1998
Torovirus-like particles related to Berne virus, BEV, and Breda virus (BRV)	Gastroenteritis	Humans, horses, and cattle	Unknown	None from biosolids	Duckmanton et al. 1997

Table E-19. Other Parasitic Pathogens of Emerging Concern

Pathogen	Disease	Source	Environmental Sources	Outbreaks Reported	Literature
Mircrosporidia	Gastroenteritis	Unknown	Unknown	None from biosolids	Johnson and Gerba 1997
Crytosporidium (Genotypes 1 and 2)	Gastroenteritis and diarrhea	Cattle	Unknown, water supply, swimming pools	None from biosolids	Patel et al. 1998, Furtado et al. 1998

Parasitic Microsporidians

Microsporidia are protozoan parasites that can infect humans and cause chronic diarrhea; they are of particular concern because of their being found in patients with AIDS (Johnson and Gerba 1997). They have only recently been discovered (seven species discovered so far) and identified as potential human pathogens, and only recent research indicates that they can be measured in environmental samples (water and wastewater) (Dowd et al. 1998). They are similar to other protozoan parasites such as *Giardia* and *Cryptosporidium* because of their small size, ability to infect different mammals, and spread through the environment; these characteristics, combined with their ability to form spores resistant to heat inactivation and drying, make them a pathogen of emerging concern with a potential to be waterborne (Johnson and Gerba 1997).

Rotaviruses

Rotaviruses are small RNA viruses that have been found to be associated with gastroenteritis in humans and a wide range of animal species (De Leon and Gerba 1990). It has yet to be shown that animal rotaviruses are pathogenic for man; furthermore, there is no evidence for species cross-infection in nature (Conklin 1981). The human rotavirus has two serotypes. Rotavirus has been associated with as many as 50% of hospitalized cases of diarrheal illness in infants and young children (EOA 1995).

Rotavirus gastroenteritis occurs worldwide both in sporadic and epidemic outbreaks. The primary targets are infants and children, particularly in the 6- to 24-month age group. Cases in adults are relatively infrequent but have been reported, mainly in countries other than the United States (EOA 1995). The most common route of rotavirus transmission is the fecal-oral route, with person-to-person transmission being the most frequent. Most individuals have acquired antibodies to both serotypes of rotavirus by the age of 2 and are therefore protected from the disease as they grow older.

In the United States, rotavirus infections are responsible for 100,000 hospitalizations per year (EOA 1995).

Rotavirus has been isolated from untreated drinking water, treated drinking water, and various foods, but the occurrence of infections from these sources has been rare (De Leon and Gerba 1990). There have been only two occurrences in the United States and these have been traced to improperly treated water (EOA 1995). No cases have been attributed to biosolids.

Rotavirus is persistent in the environment and can survive for as long as 10 days in raw fresh water and as long as 64 days in municipal treated tap water (free chlorine = 0.05 mg/l) (EOA 1995). Rotavirus has been shown to survive more than 14 days in estuarine and heavily polluted fresh water (EOA 1995). Rotavirus can survive as long as 2 weeks on inanimate surfaces, the length of survival depending on relative humidity and temperature (EOA 1995). The length of survival of rotavirus, together with its low infectious dose, leads to concerns over its possible presence in biosolids (Table 5-2 in Chapter 5). No cases of infection have been attributed to biosolids, however.

Other Viruses

Research continues to reveal the presence of previously unknown viruses that may play an important role in the large number of gastroenteritis cases of unknown origin. Among the new discoveries about which little is known are the human toroviruses (Duckmanton et al. 1997, Koopmans et al. 1997, Jamieson et al. 1998), picobirnaviruses (Gallimore et al. 1995a, 1995b; Chandra 1997), coxsachieviruses, small round structured viruses (SRSV) (Dedman et al. 1998), caliciviruses, Norwalk-like viruses (Sugieda et al. 1998), hepatitis E virus (Meng et al. 1998), Berne and Breda virus (also of animal origin), and adenoviruses. Table E-18 summarizes information on these viruses, their potential sources, and their reporting in scientific literature. Little is known about their transmission, epidemiology, environmental fate, or presence in biosolids or wastewater. However, their reporting is noted here as an indication that new pathogens continue to be discovered and that constant assessment of existing management practices is needed to ensure that biosolids are not contributing to the spread of disease. To date, no evidence indicates that they are.

Picobirnaviruses are a novel group of viruses recently found in the feces of several species of vertebrates. They have been detected in the feces of humans suffering from cryptosporidiosis and, although they have not been associated with any outbreaks attributable to water or food, are a pathogen of emerging concern. The prevalence of picnovirus in those studied in the United Kingdom was found to be 9%-13% in a wide range of patients (ages 3 to more than 65) in those both with and without the symptom of gastroentiritis (Gallimore et al. 1995b). No outbreaks caused by these viruses have been reported in the United States.

Toroviruses alone or in combination with enteroaggregative *E. coli* may play a pathogenic role in acute and possibly persistent diarrhea in children. Further studies are warranted to determine the etiologic role of toroviruses in gastroenteritis.

Other Diseases

Bovine Spongiform Encephalopathy

Well-publicized news reports in 1996 suggested that consumption of beef from diseased cattle in Britain may have caused a fatal human brain disease (Floyd 1996, Pattison 1998). The condition in the British cattle, commonly referred to as "mad cow disease" in these reports, is a disease called bovine spongiform encephalopathy, or BSE. Cattle with BSE have a degenerative brain condition that develops slowly over a 2- to 8-year period. BSE is similar in its effects on the cattle brain to other spongiform encephalopathy (SE) diseases in the brains of other animals. These include Kuru and Creutzfeldt-Jacob disease (CJD) in humans, scrapie in sheep, transmissible mink encephalopathy (TME), chronic wasting disease of mule deer and elk, feline spongiform encephalopathy (FSE), and a few others. Experimental studies have demonstrated that animals can contract some of the SE diseases by ingesting nervous system tissues (brain, spinal cord, etc.) from affected animals. It is suspected (although there is still much debate) that the causative agent in the SE diseases may be a prion, or a filterable glycoprotein devoid of detectable nucleic acid that is resistant to typical means of sterilization (Pattison 1998). These agents have survived 3 years of burial in outside soil and heating to high temperatures. An unidentified virus is also theorized as a cause.

BSE was first seen and diagnosed in Britain in 1986. It may have arisen as a result of rendered sheep byproducts being fed to cattle as protein supplements. Some of these sheep may have been infected with scrapie, an SE disease that has been known for more than 200 years. The number of BSE cases increased to a peak of about 1,000 new cases per weak by January 1993 and then began to decrease. The epidemic may have worsened because initially it was possible for cattle that had been affected with BSE to be rendered into protein supplements for other cattle. The British government banned feeding of ruminant-derived animal proteins to other ruminants in 1989. Because of the 2- to 8-year "incubation" period of development of BSE, cases continued to occur after this ban went into effect. In any event, the number of cases has decreased significantly and continues to decrease as a result of regulatory interventions, such as the offal feeding ban, which is now effectively applied.

Muscle tissue and milk have not been demonstrated to transmit BSE, but brain and spinal cord tissue have. Therefore, steps taken in Britain to ensure that nervous tissues from cattle do not enter the human food supply should effectively prevent any transmission; it is unknown whether such transmission ever actually occurred. These steps also have been taken in the United States.

To prevent the possibility of BSE entering the country, in 1989 the United States banned imports of live cattle and zoo ruminants from the United Kingdom and any country with BSE; imports of sheep and goats from the United Kingdom had already been banned because of scrapie.

No case of BSE has been diagnosed in the United States, despite aggressive efforts on the part of the U.S. Department of Agriculture and other surveillance programs for BSE. Included in the search are examinations at the National Services Veterinary Laboratory of the brains of cattle diagnosed with nervous system disease (postmortem microscopic examination of brain tissue) and periodic examinations of all live cattle in the United States that came from the United Kingdom before the import ban was instituted.

No research has been conducted to measure the presence of prions in the environment and there are no known means of measurement. Gale (1998) assessed the likelihood of prions being a risk if water from an aquifer were contaminated by a cattle-rendering plant discharging effluent to the aquifer, and found the risk of infection to be in the range of 1 in 100 million to 1 in 1 billion. Because the disease is not present in the United States, such an analysis provides further assurance that this disease represents a minimal threat to public health.

Part 2. EPA Part 503 Risk Assessment for the Land Application of Sewage Sludge

The EPA conducted extensive risk assessments for application of sewage sludge onto agricultural land and nonagricultural land (i.e., forest land, reclamation !and, and public contact sites). These assessments, based on a number of different exposure pathways and various “worst-case” (highly exposed individual or HEI) exposure assumptions, formed the basis for the sewage sludge pollutant loading limits specified in Section 503.13 of 40 CFR Part 503 Standards for the Use or Disposal of Sewage Sludge and used as minimum requirements in the SWRCB General Order (GO). The risk assessments and all the calculations and assumptions used are described in detail in technical support documents (U. S. Environmental Protection Agency 1992, Volumes 1 and 2).

Risk assessments were conducted for 14 exposure pathways for agricultural land and 12 exposure pathways for nonagricultural land. Pathway 2, human toxicity from ingesting plants grown in the home garden, and pathway 11, human exposure through inhalation of particulates resuspended by tilling of sewage sludge, were not analyzed for nonagricultural application because these are not appropriate exposure scenarios for nonagricultural land. These pathways are described in Table E-20.

The EPA assembled a national peer review committee of 35 recognized academic, government, and private industry experts in the field of sludge application to land for 10 of the risk assessments (pathways 1-10). This committee critically evaluated the methodology and data used to assess risk as part of developing criteria for land application of potentially toxic chemicals in municipal sewage sludge. The EPA's Office of Water conducted the risk assessment for pathway 11. The risk assessments for pathways 12, 13, and 14 were conducted for the EPA by the consulting firm ABT Associates (ABT Associates 1993).

Charles Henry of the University of Washington conducted thc risk assessments for pathways 1 through 10 for nonagricultural land (except for pathway 2 for home gardening). Pathways 12, 13, and 14 are identical for agricultural and nonagricultural land, so ABT Associates' assessment of agricultural pathways 12, 13, and 14 was also used for the nonagricultural pathways (U.S. Environmental Protection Agency 1992).

In undertaking the assessments, the EPA relied on numerous assumptions and decisions regarding the data to be used and what the exposure evaluations were to be based on. It was decided to use the concept of the highly exposed individual (HEI) as a target organism to be protected by the limits on individual pollutants. Depending on the pathway of exposure, the HEI could be a human, plant, animal, or environmental end point, such as surface water or groundwater, and is assumed to remain for an extended period at or adjacent to the site where the maximum exposure occurs.

Table E-20. Environmental Pathways of Concern
Identified for Application of Sewage Sludge to Agricultural Land

Pathway	Description of Highly Exposed Individual
1. Sewage Sludge-Soil-Plant-Human	Human ingesting plants grown in sewage sludge-amended soil
2. Sewage Sludge-Soil-Plant-Human	Residential home gardener
3. Sewage Sludge-Human	Children ingesting sewage sludges
4. Sewage Sludge-Soil-Plant-Animal-Human	Farm households producing a major portion of the animal products they consume; it is assumed that the animals eat plants grown in soil amended with sewage sludge
5. Sewage Sludge-Soil-Animal-Human	Farm households consuming livestock that ingest sewage sludge while grazing
6. Sewage Sludge-Soil-Plant-Animal	Livestock ingesting crops grown on sewage sludge-amended soil

Pathway	Description of Highly Exposed Individual
7. Sewage Sludge-Soil-Animal	Grazing livestock ingesting sewage sludge
8. Sewage Sludge-Soil-Plant	Plants grown in sewage sludge-amended soil
9. Sewage Sludge-Soil-Soil Organism	Soil organisms living in sewage sludge-amended soil
10. Sewage Sludge-Soil-Soil Organism-Soil Organism Predator	Animals eating soil organisms living in sewage sludge-amended soil
11. Sewage Sludge-Soil-Airborne Dust-Human	Tractor operator exposed to dust while plowing large areas of sewage sludge-amended soil
12. Sewage Sludge-Soil-Surface Water-Human	Person who consumes 0.04 kg/day of fish and 2 liters/day of water.
13. Sewage Sludge-Soil-Air-Human	Human breathing volatile pollutants from sewage sludge
14. Sewage Sludge-Soil-Groundwater-Human	Human drinking water from wells contaminated with pollutants leaching from sewage sludge-amended soil to groundwater

The risk-based models developed for the Part 503 regulations were designed to limit potential exposure of an HEI. Originally, in the 1989 proposed Part 503 rule, the concept for "worst-case" exposure was based on the "most exposed individual" (MEI), but the EPA changed this to be consistent with a statement in the rule's legislative history that calls for protecting individuals and populations that are "highly exposed to reasonably anticipated adverse conditions". In developing Subpart B of the rule, the EPA used different HEIs in evaluating each pathway of potential exposure.

The details for each of the HEIs selected and the assumptions used in the various risk scenario calculations are all contained in the technical support documents, which are voluminous (U. S. Environmental Protection Agency 1992). Examples are given here to provide an illustration of the HEIs for both the agricultural and nonagricultural settings for pathway 1, which was designed to protect consumers who eat food grown in sewage sludge-amended soil. For agricultural land application, the HEI was assumed to live in a region where a relatively high percentage of the available cropland receives sludge applications. To approximate realistic conditions, it was assumed that the HEI eats a mix of crops from land on which sludge was applied and crops from land on which sludge was not applied rather than eating foods that were all grown on sludge-amended soils.

For nonagricultural settings for pathway 1, the HEI was a person who regularly harvests edible wild plants (i.e., berries and mushrooms) from forests or rangelands that have been amended with sewage sludge. This food was assumed to be preserved by drying, freezing, or canning and, hence, to be available for consumption throughout the year. It was also assumed that an individual could continue with this practice for a lifetime (70 years).

Pathway 2 evaluated the effects on home gardeners of consuming crops grown in residential home gardens amended with sewage sludge. The major difference between pathways 1 and 2 was the fraction of food assumed to be grown on sewage sludge-amended soil. The HEI for pathway 2 was the home gardener who produced and consumed potatoes, leafy vegetables, fresh legumes, root vegetables, garden fruits (e.g., tomatoes, eggplants), sweet corn, and grains.

The HEI for pathway 3 was a young person (less than 6 year of age) ingesting sewage sludge from storage piles or from the soil surface.

For pathway 4, the HEI was an individual consuming foraging animals that consumed feed crops or vegetation grown on sewage sludge-amended soils. The HEI was assumed to consume daily quantities of the various animal tissue foods and to be exposed to background levels of pollutants from sources other than sludge. For the agricultural setting, the affected animal foods evaluated were beef, beef liver, lamb, pork, poultry, dairy, and eggs. In the nonagricultural setting, the HEI was assumed to be a hunter who preserved meat (including liver) for consumption throughout the year. The animals were assumed to have been hunted in the forest and eaten were deer and elk (because of their size and greater possibility of impact on intake through consumption compared with other animals).

Pathway 5 involved the application of sewage sludge to the land; the direct ingestion of this sewage sludge by animals; and, finally, the consumption of contaminated animal tissue by humans. The HEI was assumed to consume various animal tissue foods and be exposed to a background intake of pollutants.

Pathway 6 evaluated animals that ingest plants grown on sewage sludge-amended soil. The HEI used for both the agricultural and nonagricultural settings is a highly sensitive herbivore that consumed plants grown on sewage sludge-amended soil. Background intake was taken into account by considering background concentration of pollutants in forage crops. In a forest application site, the HEI was two grazing domestic animals and small herbivorous mammals (deer mice) that lived their entire lives in a sewage sludge-amended area feeding on seeds and small plants close to the layer of soil amended with sewage sludge. In the agricultural setting, the HEI was a sheep.

The HEI for pathway 7 was an herbivorous animal incidentally consuming sewage sludge adhering to forage crops and/or sewage sludge on the soil surface. Background intake was considered to be from ingesting soil having background levels of pollutant. Because forest animals more typically browse rather than graze, the HEI for agricultural settings was used as a reasonable worst-case surrogate for the nonagricultural HEI.

Pathway 8 was the plant phytotoxicity pathway and assumed as the HEI a plant sensitive to the pollutants in sewage sludge. Sensitivity was determined through a literature search including information on nonagronomic species, which were shown to be no more sensitive than agronomic species. Because sensitivity was found to be the same for agronomic and nonagronomic species, the limits set for agricultural species also protect wild species found in nonagricultural settings.

The HEI for pathway 9 is a soil organism sensitive to the pollutants in sewage sludge, an earthworm. Because all soil organisms are wild species, the same HEI was used for the nonagricultural and agricultural settings.

Pathway 10 assumed that the HEI was a shrew mole that consumed soil organisms that have been feeding on sewage sludge-amended soil. Pathway 9 had the same HEI for both the nonagricultural and agricultural pathways.

The HEI for pathway 11, which was designed to protect humans from the effects of airborne dusts containing sewage sludge, was a tractor driver tilling a field. This pathway evaluated the impact of particles that have been resuspended by the driver's tilling of dewatered sewage sludge into the soil. This pathway applies only to the agricultural setting because plowing is not normally performed in nonagricultural settings such as forests.

Pathway 12, the soil erosion pathway, used as an HEI a human who consumed 2 liters per day of drinking water from surface water contaminated by soil eroded from a site where sewage sludge was land applied. This individual was assumed to ingest 0.04 kilograms per day of fish from surface waters contaminated by sewage sludge pollutants. The HEI was the same for agricultural and nonagricultural practices.

Pathway 13 had as an HEI a human who inhaled the vapors of any volatile pollutants that may be in the sewage sludge when it is applied to the land. The HEI was assumed to live on the downwind side of the site with no change in wind direction ever occurring (constant exposure). The same plume air contaminant dispersion model was used for both the agricultural and nonagricultural settings.

The HEI for pathway 14 for agricultural and nonagricultural settings was an individual who obtained drinking water from ground water located directly below a field to which sewage sludge has been applied. Consumption was assumed to be 2 liters per day for a lifetime.

All the exposure scenarios involving ingestions included what is referred to as an oral reference dose (RfD). The RfD of a pollutant is a threshold below which effects adverse to human health are unlikely to occur. The EPA has a computerized listing of these human health criteria in its Integrated Risk Information System (IRIS), which it uses for many different purposes in developing health protection standards based on the latest scientific information.

Another key assumption that can change the risk assumption calculations is the recommended dietary allowances (RDAs). These are defined as the levels of intake of essential nutrients that, on the basis of scientific knowledge, are judged by the Food and Nutrition Board to be adequate to meet the known nutrient needs of practically all healthy persons. Although RfDs were generally used to determine the concentrations of inorganic pollutants that are protective of human health, the RDA was used in the case of zinc and copper.

Part 3. Endocrine Disruptors

Introduction

A wide range of chemicals, including some in common, often unregulated, undisclosed use are now associated with effects on the health, reproduction, and behavior of animals. At present, many of the effects are nonspecific in terms of the link to a particular environmental chemical, but the trends in research on hormone-affecting diseases indicate that it is probable that endocrine disruptors are contributing to human diseases and dysfunction.

The EPA has been directed by Congress to look into the issue of endocrine disruptors, focusing first on transmission in drinking water. An interagency task force of national experts has been assembled and a research plan has been developed.

Compounds termed “endocrine disruptors” can include both natural compounds and synthetic chemicals. Some, called phytoestrogens, occur naturally in a variety of plants; animals have evolved mechanisms to metabolize these, and they therefore do not accumulate and have adverse effects. A number of compounds that act as synthetic estrogens are now produced either through industrial manufacture (pesticides) or as byproducts of such processes or burning (such as dioxins). Testing for estrogenic activity is conducted in the lab using cultures of breast cancer cells. It has been found that some chemicals can cause effects at levels of parts per trillion—levels at which most chemicals have never been tested.

Table E-21 lists a variety of suspected hormone disruptors, which are discussed below.

Table E-21. List of Known and Suspected Hormone Disruptors:
Pollutants with Widespread Distribution Reported to Have Reproductive and
Endocrine-Disrupting Effects

<u>Persistent Organohalogens</u>
Dioxins and furans
PCBs
PBBsOctachlorostyrene
Hexachlorobenzene
Pentachlorophenol

<u>Pesticides</u>
2,4,5-T
2,4-D
alachlor
aldicarb
amitrole
atrazine
benomyl
beta-HCH
carbaryl
chlordane
cypermethrin
DBCP
DDT
DDT metabolites
dicofol
dieldrin
endosulfan
esfenvalerate
ethylparathion
fenvalerate
lindane
heptachlor
h-epoxide
kelthane
kepone
malathion
mancozeb
maneb
methomyl
methoxychlor
metiram
metribuzin
mirex
nitrofen
oxychlordane
permethrin
synthetic pyrethroids
toxaphene
transnonachlor
tributyltin oxide
trifluralin
vinclozolin
zineb
ziram

<u>Phenolic Compounds</u>
Penta- to Nonyl-Phenols
Bisphenol A

<u>Phthalates</u>
Di-ethylhexyl phthalate (DEHP)
Butyl benzyl phthalate (BBP)
Di-n-butyl phthalate (DBP)
Di-n-pentyl phthalate (DPP)Di-hexyl phthalate (DHP)
Di-propyl phthalate (DprP)
Dicyclohexyl phthalate (DCHP)
Diethyl phthalate (DEP)

<u>Other Organics</u>
Styrene dimers and trimers
Benzo(a)pyrene

<u>Heavy Metals</u>
Cadmium
Lead
Mercury

____

Source: Natural Resources Defense Council Endocrine Disruptors Web Page (www.nroc.org/nrdc/nrdc.proreports.html).

Pesticides

Many pesticides have been found to be estrogenic. These include the herbicides 2,4-D and 2,4,-T and the boat-fouling paint additive tributyl tin, and the traditional pesticides used widely in the past, such as carbaryl, chlordane, DDT, lindane, malathion, parathion, aldicarb, DBCP, and synthetic pyrethroids. Exposure can occur during application, through consumption of contaminated produce and other foods, through contaminated drinking water, or even from house dust in agricultural areas. Production of DDT for use in the United States was banned in 1972. However, other countries, especially tropical countries such as Mexico, still use it for mosquito control to combat malaria. DDT and its metabolites bioaccumulate in wildlife, and humans can be exposed through the food chain.

Soaps, Shampoos, and Hair Colors

Many industrial and consumer products contain alkylphenol ethoxylates (APEs), which break down into alkylphenols such as nonylphenol, which has been found in sewage and rivers near outfalls. One of the main uses of these compounds is in liquid detergents. In Europe, these products have been replaced by the more expensive but much safer alcohol ethoxylates. Denmark based its phaseout of alkyphenol exthoxylate on research conducted in the United Kingdom, which found that its breakdown products, alkylphenols, caused male fish to take on female characteristics. Alkylphenols do not biodegrade easily and bioaccumulate and therefore may cause problems when sewage sludge is applied to land.

Plastics and Plasticizers

Plastics contain additives, such as phthalates, bisphenol-A, and nonylphenols, usually present as plasticizers to increase flexibility and durability. They can leach out into liquids and foods. Heating speeds up this leaching process, which is why microwaving of foods in plastic is discouraged. Estrogenic butyl benzyl phthalate is found in vinyl floor tiles, adhesives, and synthetic leathers. The related compound di-butyl phthalate is present in some food-contact papers. Bisphenol-A is a breakdown product of polycarbonate plastics, which are used in water bottles, baby bottles, and the linings of some food cans.

Polychlorinated Biphenyls (PCBs)

PCBs are a family of toxic industrial chemicals commercialized in 1929 by Monsanto. Although their production in the United States stopped in 1977, world production continued. PCBs are still present in the United States in electrical equipment and are frequently found at toxic waste sites and in contaminated sediments. A recent study confirmed that children exposed to low levels of PCBs in the womb because of their mother's fish consumption grow up with low IQs, poor reading comprehension, difficulty paying attention, and memory problems.

Dioxins

Chlorinated dioxins and dibenzofurans are byproducts of the chlorine bleaching of paper; the burning of chlorinated hydrocarbons such as pentachlorophenol, PCBs, and polyvinyl chloride; the incineration of municipal and medical wastes; and natural events, such as forest fires and volcanic eruptions. They often contaminate toxic wastes sites, especially where there have been fires. They bioaccumulate in fish and other wildlife, and the most common human route of exposure is through the food chain.

Spermicides

Many spermicides contain nonoxynol-9, a nonylphenol that kills sperm. This compound can be carried into the sewer system and hence into biosolids, although the concentrations are probably not measurable.

Preservatives

BHA, butylated hydroxyanisole, is added to foods such as breakfast cereal, or its packaging, to prevent the foods from becoming rancid.

Metals

Lead, methyl mercury, and cadmium can disrupt the endocrine system by causing problems in steroid production.

In addition, a number of other pollutants with widespread distribution in the environment are reported to bind to hormone receptors and therefore are suspected to have reproductive and endocrine-disrupting effects. These pollutants include the following:

- 2,4-dichlorophenol
- diethylhexyl adipate
- benzophenone
- N-butyl benzene
- 4-nitrotoluene

The compounds listed above are only suspected of being endocrine disruptors. All of these compounds have had wide uses in the past and are present in the environment, although only a few are likely to be found. Their presence in biosolids, soils, water, food, or animals is variable and depends on the historical use of the chemicals and the means of environmental distribution. At present, there is no evidence that their presence in biosolids would increase health risks.

Citations

Printed References

ABT Associates, Inc. 1993. Human health risk assessment for the use and disposal of sewage sludge: benefits of regulation. Government Reports Announcements & Index (GRA&I) 24. Cambridge, MA.

Ansari, S. A., S. R. Farrah, and G. R. Chaudhry. 1992. Presence of human immunodeficiency virus nucleic acids in wastewater and their detection by polymerase chain reaction. Applied and Environmental Microbiology 58:3984-3990.

Argent, V. A., J. C. Bell, and D. Edgar. 1981. Animal disease hazards of sewage-sludge disposal to land: effects of sludge treatment on Salmonellae. Water Pollution Control 80:537-540.

Argent, V. A., J. C. Bell, and M. Emslie-Smith. 1977. Animal disease hazards of sludge disposal to land: occurrence of pathogenic organisms. Water Pollution Control 76:511-516.

Belongia, E. A., T. T. Osterholm, J. T. Soler, D. A. Arumend, J. E. Braun, M. D. MacDonald. 1993. Transmission of *Escherichia coli 0157:H7* infection in Minnesota child day-care facilities. JAMA 269(7):883-888.

Blostein, J. 1991. Shigellosis from swimming in a park pond in Michigan. Public Health Reports 106(3):317-322.

Boutin, P., M. Torre, and J. Molina. 1987. Bacterial and fungal atmospheric contamination at refuse composting plants: a preliminary study. In de Bertoldi, M., M.P. Ferranti, P. L'Hermite, F. Zucconi, Compost: production, quality and use. Elsevier Applied Science, for Commission of the European Communities. The Hague, Netherlands.

Cascio, A., M. Bosco, E. Vizzi, A. Giammanoco, D. Ferraro, S. and Arista. 1996. Identification of picobirnavirus from faeces of Italian children suffering from acute diarrhea. European Journal of Epidemiology 12(5):545-547.

Casson, L.W., C. A. Sorber, R. H. Palmer, A. Enrico, and P. Gupta. 1992. HIV survivability in wastewater. Water Environmental Research 64:213-215.

Centers for Disease Control. 1993. Multistate outbreak of Escherichia coli 0157:H7 infections from hamburgers—western United States, 1992-1993. MMWR 42(14):259-262.

__________. 1999. Internet home page containing Emerging Diseases Information and Morbidity and Mortality Weekly Reports. U.S. Department of Health and Human Services, Centers for Disease Control. Www.cdc.gov/cdctext.htm. February 8-12, 1999.

Chandra, R. 1997. Picobirnavirus, a novel group of undescribed viruses of mammals and birds: a minireview. Acta Virol. 41(1):59-62.

Cieslak, P. R., K. F. Gensheimer, et al. 1992. *Escherichia coli 0157:H7* infection from a manured garden. The Lancet 342:367.

Clark, C. S., R. Rylander, and L. Larson. 1983. Levels of gram-negative bacteria, *Aspergillus fumigatus*, dust and endotoxin at compost plants. Applied and Environmental Microbiology 45:1501-1505.

Conklin, R. H. 1981. Rotavirus infections; CRC handbook series in zoonoses. CRC Press. Boca Raton, FL.

Cravaghan, P. D., et al. 1993. Inactivation of *Giardia* by anaerobic digestion of sludge. Water Science Technology 27:111.

De Leon, R., and C. P. Gerba. 1990. Detection of rotaviruses in water by gene probes. Wat. Sci. Tech. 24(2):281-284.

Dedman D., H. Laurichesse, E. O. Caul, P. G. Wall. 1998. Surveillance of small round structured virus (SRSV) infection in England and Wales, 1990-5. Epidemiolinfect 121(1):139-149.

Diaz, L. F., G. M. Savage, L. C. Eggerth, and C. B. Golueke. 1992. Composting and recycling municipal solid waste. Lewis Publishers. Boca Raton, FL.

Dowd, S. E., C. P. Gerba, and I. L. Pepper. 1998. Confirmation of the human-pathogenic microsporidia *Enterocytozoon bieneusi*, *Encephalitozoon intestinalis*, and *Vittaforma corneae* in water. Applied and Environmental Microbiology 64(9):3332-3335.

Droffner M. L., and W. F. Brinton. 1995. Survival of *E. coli* and *Salmonella* populations in aerobic thermophilic composts as measured with DNA gene probes. Zentralbl Hyg Umweltmed 197(5):387-397.

Duckmanton L., B. Luan, J. Devenish, R. Tellier, and M. Petric. 1997. Characterization of torovirus from human fecal specimens. Virology 239(1):158-168.

Dunaway, W. C., et al. 1983. 1982 Shigellosis outbreak in Omaha, Nebraska. Nebraska Medical Journal 68(6):165-168.

Enriques, C. E., C. J. Hurst, and C. P. Gerba. 1995. Survival of the enteric adenoviruses 40 and 41 in tap, sea and wastewater. Wat. Res. 29(11):2548-2553.

EOA, Inc. 1995. Microbial risk assessment for reclaimed water. Final report. May 10, 1995. Prepared for Irvine Ranch Water District. Prepared in Association with the University of California School of Public Health.

Epstein, E. 1993. Neighborhood and worker protection for composting facilities: issues and actions. In Hoitink, H. A. J., and H. M. Keener (eds.), Science and engineering of composting: design. Environmental, microbiological and utilization aspects. Renaissance Publications, for Ohio State University. Worthington, OH.

Epstein, E., and J. I. Epstein. 1985. Health risks of composting. Biocycle 26(4):38-40.

__________. 1989. Public health issues and composting. Pages 120-124 in Epstein, E., and J. I. Epstein (eds.), The biocycle guide to yard waste composting. J.G. Press. Emmaus, PA.

Evans, M. R. 1998. *Salmonella enteritidis* PT6: another egg-associated salmonellosis? Emerging Infectious Diseases 4(4):667-669. Centers for Disease Control.

Feachem, R. G., et al. 1980. Appropriate technology for water supply and sanitation. December. World Bank.

__________. 1983. Sanitation and disease: health aspects of excreta and wastewater management. John Wiley and Sons. New York, NY.

Floyd, J. 1996. Don't call it "mad cow disease" or bovine spongiform encephalopathy (BSE) just the facts. Extension Veterinarian, Auburn University [Alabama]. March 29, 1996.

Furtado C., G. K. Adak, J. M. Stuart, P. G. Wall, H. S. Evans, and D. P. Casemore. 1998. Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992-5. Epidemiol. Infect. 121 (1):109-119

Gale, P. 1998. Quantitative BSE risk assessment: relating exposures to risk. Letters in Applied Microbiology 27:239-242.

Gallimore, C. I., H. Appleton, D. Lewis, J. Green, and D. W. Brown. 1995a. Detection and characterization of bisegmented double-stranded RNA viruses (picobirnaviruses) in human fecal specimens. J. Med. Virol. 45(2):135-140.

Gallimore, C. I., J. Green, D. P. Casemore, and D. W. Brown. 1995b. Detection of a picobirnavirus associated with *Cryptosporidium* positive stools from humans. Archives of Virology 140(7):1275-1278.

Hammerberg, B., G. A. MacInnis, and T. Hyler. 1978. *Taenia saginata cysticerci* in grazing steers in Virginia. J. Am. Vet. Med. Assoc. 173:1462-1464.

Hanninen, M. L., and A. Siitonen. 1995. Distribution of Aeromonas phenospecies and genospecies among strains isolated from water, foods or from human clinical samples. Epidemiol. lnfect. 115(1):39-50.

Hu, C. J., R. A. Gibbs, N. R. Mort, H. T. Hofstede, G. E. Ho, and I. Unkovich. 1996. *Giardia* and its implications for sludge disposal. Pages 179-186 in Water Science and Technology, 1996. Water Quality International, '96. Part 4. Edited by Ballay, D., et al. Proceedings of the 18th Biennial conference of the International Association on Water Quality, Singapore, June 23-28, 1996.

Hulten K., H. Enroth, T. Nystrom, and L. Engstrand. 1998. Presence of Helicobacter species DNA in Swedish water. J App1. Microbiol. 85(2):282-286.

Jamieson F. B., E. E. Wang, C. Bain, J. Good, L. Duckmanton, and M. Petric. 1998. Human torovirus: a new nosocomial gastrointestinal pathogen J. lnfect. Dis.178(5):1263-1269.

Joce, R. E., et al. 1991. An outbreak of cryptosporidiosis associated with a swimming pool. Epidemiol. Infect. 107(3):497-508.

Johnson, D. C., and C. P. Gerba. 1997. Microsporidia - the next cryptosporidium? Water Conditioning and Purification. September:116-119.

Jones I. G., and M. Roworth. 1996. An outbreak of *Escherichia coli 0157* and campylobacteriosis associated with contamination of a drinking water supply. Public Health 110(5):277-82.

Jones, P. W., L. M. Rennison, V. H. Lewin, and D. L. Redhead. 1980. The occurrence and significance to animal health of salmonellas in sewage and sewage sludges. J. Hyg. 84:47-62.

Kinde, H., M. Adelson, A. Ardans, H. E. Little, D. Willoughy, D. Berchold, D. H. Read, R. Breitmeyer, D. Kerr, R. Tarbell, and E. Hughs. 1997. Prevalence of salmonella in municipal sewage treatment plant effluents in southern California. Avian Diseases 41(2):392-398.

Kinde, H., D. H. Read, R. P. Chin, A. A. Bickeford, R. L. Walker, A. Ardans, H. E. Little, D. Willoughy, D. Berchold, R. Breitmeyer, D. Kerr, and I. A. Gardner. 1996. *Salmonella enteritidis,* phase type 4 infection in a commercial laying flock in southern California: bacteriological and epidemiologic findings. Avian Diseases 40(4):665-671.

Koopmans, M. P, E. S. Goosen, A. A. Lima, I. T. McAuliffe, J. P. Nataro, L. J. Barrett, R. I. Glass, and R. L. Guerrant. 1997. Association of torovirus with acute and persistent diarrhea in children Pediatr. Infect. Dis. 16(5):504-507.

Koutkia, P., E. Mylonakis, and T. Flanigan. 1997. Enterohemorrhagic *Escherichia coli* O157:H7 - an emerging pathogen. Am. Fam. Physician 56(3):853-856, 859-861.

Ludert, J. E., L. Abdul-Latiff, A. Liprandi, and F. Liprandi. 1995. Identification of picobirnavirus, viruses with bisegmented double stranded RNA, in rabbit faeces. Res. Vet. Sci. 59(3):222-225.

Maritato, M. C., E. R. Algeo, and R. E. Keenan. 1992. The *Aspergillus fumigatus* debate: potential human health concerns. BioCycle 13:70-72.

McKenzie, W. R. et al. 1994. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. The New England Journal of Medicine331(3):161-167.

Meng, X. J., P. G. Halbur, J. S. Haynes, T. S. Tsareva, J. D. Bruna, R. L. Royer, R. Purcell, and S. U. Emerson. 1998. Experimental infection of pigs with the newly identified swine hepatitis E virus (swine HEV), but not with human strains of HEV. Arch. Virol. 143(7):1405-1415.

Millner, P. D., P. B. Marsh, R. B. Snowden, and J. F. Parr. 1977. Occurrence of *Aspergillus fumigatus* during composting of sewage sludge. Applied and Environmental Microbiology 34:765-772.

Millner, P. D., D. A. Bassatt, and P. B. Marsh. 1980. Dispersal of *Aspergillus fumigatus* from sewage sludge compost piles subjected to mechanical agitation in open air. Applied and Environmental Microbiology 39:1000-1009.

Mishu B., J. Koehler, L. A. Lee, D. Rodrigue, F. H. Brenner, P. Blake, et al. 1994. Outbreaks of *Salmonella enteritidis* infections in the United States, 1985-1991. Journal of Infect. Dis.169:547-52.

Moore, A. C., et al. 1993. Surveillance for waterborne disease outbreaks—United States, 1991-1992. Morbidity and Mortality Weekly Report 42:SS-5, 1-22.

Moore, B. E. 1993. Survival of human immunodeficiency virus (HIV), HIV-infected lymphocytes, and poliovirus in water. Applied and Environmental Microbiology 59:1437-1443.

Morris R. D, E. N. Naumova, J. K. Griffiths. 1998. Did Milwaukee experience waterborne cryptosporidiosis before the large documented outbreak in 1993. Epidemiology 9(3):264-70.

Nelson, H. 1997. The contamination of organic produce by human pathogens in animal manures . EAP Publications.

Olver,W. M. 1979. The life and times of *Aspergillus fumigatus*. Compost Science/Land Utilization 202:36-39.

Padhye, N. V., and P. Doyle. 1992. *Escherichia coli 0157:H7*: epidemiology, pathogenesis, and methods for detection in food. Journal of Food Protection 55(7):555-565.

Patel, S., S. Pedraza-Diaz, J. McLauchlin, and D. P. Casemore. 1998. Molecular characterization of Cryptosporidium parvum from two large suspected waterborne outbreaks. Outbreak Control Team South and West Devon 1995, Incident Management Team and Further Epidemiological and Microbiological Studies Subgroup North Thames. Commun. Dis. Public Health 1(4):231-233.

Pattison, J. 1998. The emergence of bovine spongiform encephalopathy and related diseases. Emerging Infectious Diseases 4(3):390-394.

Preston, D. R., S. R. Farrah, G. Bitton, and G. R. Chaudhry. 1991. Detection of nucleic acids homologous to human immunodeficiency virus in wastewater. Journal of Virological Methods 33:383-390.

Raper, K. B., and D. I. Fennel. 1965. The genus *Aspergillus*. Williams and Wilkins Co. Baltimore, MD.

Robertson, L. J., and H. V. Smith. 1992. *Cryptosporidium* and cryptosporidiosis; part 1: current perspective and present technologies. European Microbiology 1992:20-29.

Roivainen, M., M. Knip, H. Hyoty, P. Kulmala, M. Hiitunen, P. Vahasalo, T. Hovi, and H. K. Akerblom. 1998. Several different enterovirus serotypes can be associated with prediabetic autoimmune episodes and onset of overt IDDM. Childhood Diabetes in Finland (DiMe) Study Group. J. Med. Virol. 56 (1):74-78.

Rosenberg, A. S., and G. Y. Minimato. 1996. Aspergillosis in AIDS. The AIDS Reader 6(5):173-178.

Singh V., V. Singh, M. Raje, C. K. Nain, and K. Singh. 1998. Routes of transmission in the hepatitis E epidemic of Saharanpur. Trop. Gastroenterol.19(3):107-109.

Sinski, J. T. 1975. The epidemiology of aspergillosis. In Al-Doory, Y. (ed)., The Epidemio1ogy of Human Mycotic Diseases. Charles C. Thomas Co. Springfield, IL.

Sorvillo, F. J., et al. 1988. Shigellosis associated with recreational water contact in Los Angeles County. Am. J. Trop. Med. 38(3):613-7.

__________. 1992. Swimming-associated cryptosporidiosis. American Journal of Public Health 82(5):742-744.

St. Louis, M. E., D. L. Morse, M. E. Potter, T. M. De Melfi, J. J. Guzewich, R. V. Tauxe, et al. 1988. The emergence of grade A eggs as a major source of *Salmonella enteritidis* infections. JAMA 259:2103-2107.

Straub, T. M., I. L. Pepper, and C. P. Gerba. 1993. Hazards from pathogenic microorganisms in land-disposed sewage sludge. Reviews of Environmental Contamination and Toxicology 132:58-61.

Sugieda M., H. Nagaoka, Y. Kakishima, T. Ohshita, S. Nakamura, and S. Nakajima. 1998. Detection of Norwalk-like virus genes in the caecum contents of pigs. Arch. Virol. 143(6):1215-1221.

Tzipori, S. 1988. Cryptosporidiosis in perspective. Adv. Parasitol. 27:63-129.

U.S. Environmental Protection Agency. 1985. Pathogen risk assessment feasibility study. Office of Research and Development, Cincinnati, OH, and Office of Water Regulations and Standards, Washington, DC.

__________. 1992. Technical support document for reduction of pathogens and vector attraction in sewage sludge. Office of Water. (WH-586.) EPA 822/R-93-004. November.

Wadstrom, T., and A. Ljungh. 1991. *Aeromonas* and *Plesiomonas* as food- and waterborne pathogens. Int. Food Microbiol. 12(4):303-11.

Wells, J. G., L. D. Shipman, K. D. Greene, E. G. Sowers, J. H. Green, D. N. Cameron, F. P. Downes, M. L. Martin, P. M. Griffin, S. M. Ostroff, M. E. Potter, R. V. Tauxe, and I. K. Wachsmuth. 1991. Isolation of *Escherichia coli* serotype 0157:H7 and other shiga-like-toxin-producing *E. coli* from dairy cattle. Journal of Clinical Microbiology 29(5):985-989.

Whitmore, T. N., and L. J. Robertson. 1995. The effect of sewage sludge treatment processes on oocysts of *Cryptosporidium paryum.* Journal of Applied Bacteriology 78(1):34-38.

Personal Communications

Cook, Raymond. Registered environmental health specialist. Kings County Health Department, Hanford, CA. February 1, 1999 - telephone conversation.

Gerba, Charles, Ph.D. Professor. University of Arizona, Phoenix, AZ. February 16, 1999 - telephone communication.

Shaw, Guy. Environmental health specialist. Kern County Health Department, Bakersfield, CA. February 3, 1999 - telephone conversation and facsimile transmission.

Starr, Dr. Mark. California Department of Health Services Disease Investigations & Surveillance Branch, Surveillance & Statistics Section. February 2, 1999 - telephone conversation and electronic data transmittal.